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BUNDLE BRANCH BLOCK

Historical and Experimental

Block of either or both of the main branches of the auriculoventricular bundle of His (bundle branch block) was identified early in the history of clinical electrocardiography. This condition has been the source of much study because of the bizarre electrocardiographic forms that it produces and because of the serious clinical conditions with which it is usually associated. Faint, slurred or split first heart sound and split second sound are generally observed with bundle branch block; but these are not specific diagnostic signs. The electrocardiogram is the sole certain means for making a diagnosis.

Eppinger and Rothberger, in 1910, sectioned separately the right and left main branches of the auriculoventricular bundle in dogs, and produced the forms of deflections which they termed "levocardiograms" and "dextrocardiograms," respectively. Later (1916) Lewis obtained these forms by similar sections of the hearts of large rhesus monkeys.

Attention was further drawn to this condition when corresponding curves were found in the electrocardiograms of human beings with serious cardiac disease. Mathewson (1912), Lewis (1913) and Carter (1914) all assigned these specific aberrations of form to interruption of the same branch of the auriculoventricular bundle as that which produced the characteristic records in experimental bundle branch block of dogs. In man the levocardiogram type was found over ten times more frequently than the dextrocardiogram type.

Carter's criteria for what will henceforth be termed the "classic form" of bundle branch block were: (1) widening of the QRS complex beyond 0.1 second, with notching; (2) preponderance of the ventricle supplied by the uninterrupted bundle branch, either right or left axis deviation with dextrocardiogram appearing in left bundle branch block, and the levocardiogram in right bundle branch block; (3) exaggeration of the amplitude of all the ventricular deflections including the T wave (a criterion now generally discarded); and (4) the T wave in an opposite direction to the chief portion of the QRS deflection (recently used as evidence, though not as yet accepted, of intraventricular conduction system defects in the absence of other characteristics).

Failure to find the expected lesions by pathologic examination first raised the doubt as to the validity of diagnosing the particular branches to which the lesions in man were assigned by comparison with records obtained in experiments on animals. Fahr raised objection also on theoretic grounds to this early teminology.

These doubts have led to the general acceptance recently of the exact reverse of the original conception of the dextrocardiogram and levocardiogram in man, — namely, that in the common type, or left bundle branch block (dextrocardiogram), the initial ventricular deflections are upright in Lead I and downward in Lead III; while in the infrequent type, or right bundle branch block (levocardiogram), the chief deflection is downward in Lead I and upright in Lead III. Lead IV, the chest lead, gives records that closely resemble those of Lead I, using the revised IV F lead method of placing the electrodes (RA on the left leg and LA over the apex of the heart).

Evidence for this reversal of terminology has been obtained from work on human hearts. Barker, MacLeod and Alexander, and Wilson, found that artificially-produced ectopic beats in various portions of either ventricle of an exposed human heart produced electrocardiograms that were approximately reverse in form to those produced in dogs by Kraus and Nicolai, Lewis, and others. There has been much experimental work done recently that tends to confirm these conclusions, especially that of Kountz, Prinzmetal, Pearson and Koenig on revived human hearts.

It must be remembered that there are many factors determining the electrical axis of the heart and therefore the direction of the chief ventricular deflection in Leads I and III. However, for the practical purpose of uniformity, it is probably wise to adhere to the new terminology although there is still some controversy existing, especially in Europe, regarding the assignment of the location of these blocks.

Variations in Form —"Intraventricular Block"

There has been much discussion concerning the limitation of the term "bundle branch block" to those instances presenting the unmodified criteria of Carter.

The curves upon which the greatest emphasis has been placed are those described by Wilson and his associates: (1) resembling right bundle branch block but with a sharp high R wave preceding a slurred S wave in Lead I; and (2) with a slurred deep S_1 and flattened or upright T_1 associated with the major QRS deflection downward and upright T waves in Leads II and III, as would be expected in left bundle branch block. From precordial leads, these were believed to represent right bundle branch lesions. There is a common tendency to recognize all cases of slurring and widening of QRS complexes over 0.1 second in any or all leads that cannot be classified in any of the categories mentioned above, as atypical bundle branch blocks, and to term them intraventricular blocks.

Herrmann and Ashman and others have shown that damage to the terminal ramifications of the auriculoventricular bundle practically never produces delay in the description of the QRS complex. Partial or complete obstruction of a main branch of the auriculoventricular bundle is necessary to produce this picture.

Transient, Multiple and Variable Forms

Transient bundle branch block has frequently been reported, as well as instances of shift from block of one branch to block of the other. Likewise there often occurs gradual development of the classic forms from the normal electrocardiogram, or intermediate curves and similar slow recessions. Such changes, both gradual and abrupt, tend to be pro-

duced by a variable degree of myocardial anoxia such as occurs in congestive heart failure, paroxysmal tachycardia, auricular flutter or auricular fibrillation, general anesthesia, shock, pulmonary arterial thrombosis, and faulty pulmonary ventilation as in pneumonia. Development of edema in the region of the conduction system, such as occurs in congestive failure or local infections, and the local action of certain toxic substances such as occurs in uremia and bacterial infections are reported as causes of these fluctuating blocks.

One is at a loss whether or not to interpret single records presenting intermediate forms as partial or impending bundle branch block. There are apparently no existing criteria that aid definitely in reaching a decision. It is hoped that the monocardiograms (vectorcardiograms), a summated form of the electrocardiogram obtained by Mann and by Wilson and Johnston using the cathode ray oscillograph, may furnish diagnostic clues that cannot be found in records taken by the customary method.

There is a clinical entity that was first described by Wolff, Parkinson and White, and that has been observed with minor variations by Tung, Pines and others. The condition is usually found in otherwise normal individuals who have a tendency to paroxysmal tachycardia or auricular fibrillation. Their electrocardiograms show the curves of a classic bundle branch block with short P-R intervals during their "normal" periods, and normal ventricular complexes during the attacks of paroxysmal tachycardia.

Anatomy and Pathology

It is now well recognized, from studies made by Eppinger and Stoerk, Cohn and Lewis, and more recently by Mahaim, Oppenheimer and his associates, and Yater, that lesions are almost invariably present in both branches of the auriculoventricular bundle when either right or left bundle branch block is found by the patient's electrocardiograms. Masters, as well as Yater, has found that either the anterior perforating arteries from the left anterior descending coronary artery or the first posterior perforating artery from the right coronary artery, may be involved in the production of lesions in this region, the latter being somewhat more frequently involved.

The association of bundle branch lesions with partial or complete auriculoventricular heart block frequently occurs. This auriculoventricular block has been ascribed in several cases to bilateral bundle branch block, but in the concomitant existence of auriculoventricular and bundle branch block would be expected because of a joint arterial supply. The auriculoventricular node, the common portion of the auriculoventricular bundle, the first portions of both branches, and the posterior extension of the lower left branch are generally supplied by the first posterior perforating branch of the right coronary artery. The main portion of the right bundle branch is generally supplied by the perforating twigs of the left anterior descending coronary artery, as is also the lower anterior portion of the left branch of the auriculoventricular bundle branch. Anastomoses usually occur between these perforating branches of the left and right coronary artery, but are not thought to function adequately in most instances of sudden occlusion of either artery.

Etiology

As might be expected, atherosclerosis of the arteries, and arteritis or direct damage of the conduction system tissue by syphilis or the unknown agent of the acute rheumatic syndrome, are the commonest causes of bundle branch as well as of partial and of complete auriculoventricular block. The condition occurs much more frequently after than before the age of 45, although the atypial or "intraventricular" blocks are relatively more common before than after this age and are therefore more commonly due to other etiological agents than atherosclerosis.

Cardiac developmental defects, especially of the intraventricular septum, often produce aberrations of the QRS complexes, but it has been the author's experience that in these instances the electrocardiogram very rarely assumes the classic bundle branch block form. Pressure on the conduction system by tumors or aneurysm have been reported as a rare cause of block. The occurrence of bundle branch block, both permanent and transient, has been reported in patients with diphtheria and in others with thyrotoxicosis.

It has been believed by several investigators that those cardiac lesions such as hypertension that cause strain on the left ventricle tend to produce left bundle branch block; and that, conversely, lesions such as rheumatic mitral stenosis that cause right ventricular hypertrophy tend to produce right bundle branch block. Statistically, there may be some evidence supporting this concept; but both forms of block seem to occur too frequently in all etiologic conditions to warrant the conclusion that the casual relationship is more than suggestive.

Acute coronary occlusion has been recognized as a frequent cause of intraventricular conduction system block. Masters states that 15 per cent of all such lesions are associated with these electrocardiographic changes. In the author's experience, the classic forms of bundle branch block are not found as frequently as is the atypical intraventricular block. Since it is now known that coronary artery occlusion may develop without pain, the otherwise unexplained appearance of intraventricular block should suggest the existence of this condition.

Prognosis

It is recognized that partial or complete bundle branch block may occur from involvement of only one small arterial branch or from a small focal lesion of the conduction tissue. Such an electrocardiographic abnormality does not necessarily signify serious generalized cardiac disease. It is not remarkable therefore that many patients with permanent bundle branch block have been reported who were in reasonably good health five, ten or even fifteen years after the discovery of the lesion. The prognosis of the atypical forms of bundle branch block (intraventricular block), including the type described by Wilson and his associates, was found, in the experience of Nagle and the author, to be considerably better than that of the classic form. In the entire group of patients with atypical forms there was a fatality of 40+ per cent, as contrasted with 75+ per cent in the group with the classic form. Masters indicated the increased gravity of acute coronary arterial occlusion when bundle branch occurs by showing that the fatality rate of such cases was 42 per cent, as contrasted to the presently accepted fatality rate of all cases of acute coronary arterial occlusion, which is 15 to 30 per cent.

Most investigators have found that the majority of the patients with these lesions who died, did so within the first year after the discovery of their bundle branch blocks. Nagle and I evaluated the prognosis in both the classic and atypical forms; and found that the chance of survival to a normal life expectancy was approximately 40 per cent greater in individuals surviving the first year after the discovery of the lesion, that it nearly doubled in those surviving the second year, and that these conduction system lesions alone had practically no influence on life expectancy after the fifth year of survival following the discovery of the lesions.

In spite of the possibility that such conduction system lesions may be small focal involvements, the assumption is usually made that they represent objective evidences of general cardiac diseases, most probably widespread atherosclerosis of the coronary arterial tree. Unless the condition is proved to be of another nature, the occurrence of these electrocardiographic abnormalities must be taken as a serious prognostic sign.

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